

Complete resection of pulmonary inflammatory pseudotumors has excellent long-term prognosis

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Objective: Pulmonary inflammatory pseudotumor is an uncommon disease, often with a benign presentation. However, invasion of adjacent thoracic organs, local recurrence, and distant metastases have been described, and the best management strategy remains unclear. We present a single large institutional experience in patients with pulmonary inflammatory pseudotumor and propose guidelines for treatment of this patient population.

Methods: A retrospective study was performed to review all patients who underwent resection for pulmonary inflammatory pseudotumor between 1974 and 2007.

Results: A total of 25 patients were treated with pulmonary inflammatory pseudotumor at the Marie Lannelongue Hospital. The mean age was 33 years. Two patients were referred after an incomplete resection. One patient presented with cerebral metastasis. We performed a complete resection in all patients: wedge resection ($n = 7$), lobectomy ($n = 6$), sleeve arterial lobectomy ($n = 1$), lobectomy with thoracic inlet exenteration ($n = 2$), bilobectomy ($n = 2$), pneumonectomy with brain metastasectomy ($n = 1$), sleeve pneumonectomy ($n = 2$), sleeve main bronchus or tracheal resection ($n = 2$), wedge with sleeve main pulmonary artery resections ($n = 1$), and sleeve pneumonectomy with esophageal, aortic arch, and right pulmonary artery resection ($n = 1$). No adjuvant therapy was given to any patients. Postoperative 30-day mortality and morbidity rates were 4% and 8%, respectively. With a mean follow-up of 80 months (range 4–369 months, 100% follow-up), actuarial 10-year survival was 89%. One patient died of an extensive sarcomatous recurrence 2 years after surgery.

Conclusion: Pulmonary inflammatory pseudotumor is a malignant disease affecting young patients with local invasion, distant metastasis, local recurrence, and sarcomatous degeneration. A complete resection should always be performed at initial presentation because of its high likelihood of cure with aggressive management.

Inflammatory pseudotumor (IPT) of the lung is a rare and not well-described disease. This disease has presented in the larynx, retroperitoneum, and abdomen. The lung is the most common site of localization, accounting for 0.7% of all thoracic tumors.^{1–12} In children aged less than 16 years, IPT is the most frequent primary pulmonary tumor.¹³ Although IPT is usually considered to be a benign entity, local invasion, local postoperative recurrence, and distant metastases have been described. Its histologic presentation and natural history are widely variable; therefore, a variety of terms have been used to identify this disease, including plasma cell granuloma,⁷ inflammatory myofibroblastic tumor,¹² xanthogranuloma,¹⁴ or fibrous histiocytoma.⁵ Because of this inconsistency in pathologic diagnosis and the small number of patients typically seen with this disease, the treatment of choice remains debated.

At the Marie Lannelongue Hospital, we have approached this disease as a low-grade sarcoma. Therefore, it has been our policy to perform a complete R0 resection whenever possible. Patients with pulmonary IPT are typically young; therefore, an aggressive approach is warranted to maximize the likelihood of long-term cure. We retrospectively reviewed our single-center experience to determine the effectiveness of complete resection of pulmonary IPT.

MATERIALS AND METHODS

We performed a chart review of all patients who presented to the Marie Lannelongue Hospital between 1974 and 2007. We identified 26 patients with pulmonary IPT. One patient was not deemed suitable for surgery because of a bulky tumor invading the esophagus, descending aorta, and left atrium, which was associated with poor conditions that precluded extended surgery. This patient was treated medically by imatinib and steroids. A lethal aortoesophageal fistula developed in the patient 6 months later, and he was excluded from the present study. All the remaining 25 patients underwent surgery in our department. This patient population accounted for 0.05% of all thoracic surgical procedures performed during this period. Records of the patients with pulmonary IPT were reviewed, and the following data were collected: clinical presentation, medical history, laboratory data, and radiologic findings at the time of diagnosis, diagnostic procedure, surgical treatment, postoperative complications, and long-term follow-up. The study was approved by our institutional review board.

The preoperative workup included physical examination, chest radiography, computed tomography of the chest and upper abdomen, computed tomography or magnetic resonance imaging of the brain, bronchoscopy, spirometry, arterial blood gas measurement, ventilation-perfusion scanning, and electrocardiography. F-18 fluorodeoxyglucose positron emission

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Abbreviation and Acronym

IPT = inflammatory pseudotumor

tomography was performed for only recent cases ($n = 2$) and demonstrated a lesion with intense F-18 fluorodeoxyglucose activity with a standardized uptake value of 10 and 8, respectively. In patients at high risk, a right-sided heart catheterization was performed before and after balloon occlusion of the relevant pulmonary artery to detect pulmonary hypertension.

Tumor resection was considered complete when the resection margins were free of disease. Two pathologists (V.M. and E.D.) independently reviewed the histologic specimens of all cases to confirm the diagnosis of IPT. Immunophenotyping was determined by immunohistochemical staining on 5- μ m-thick, formalin-fixed, paraffin-embedded tissue sections, and clonality was characterized according to the type of immunoglobulin on malignant cell surface in all cases. Tissue sections were incubated with monoclonal antibodies 1) to cytokeratins (AE 1–3 clone; Dako, Carpinteria, Calif; 1:125) to rule out a spindle cell carcinoma, 2) to smooth muscle actin (1A4 clone; Dako; 1:100), 3) to anaplastic lymphoma kinase-1 (Dako; 1:100), and 4) to CD34 and S100 protein.

Tumors were classified according to the criteria defined by Gal and colleagues² and the World Health Organization.¹⁵ The histologic type defined by Matsubara and colleagues¹⁶ as organizing pneumonia was excluded from this review because of its non-neoplastic nature. Follow-up data were obtained by telephone or patient's hospital visit.

Determination of long-term follow-up was calculated from the date of diagnosis to the date of death or the last follow-up visit. Continuous data are presented as mean \pm standard error unless otherwise noted. Survivals were

calculated by life-table analysis with the date of resection of the IPT as the starting time. Kaplan–Meier curves were plotted and compared using the log-rank test for univariate analysis with StatView V (Abacus Concepts, Berkeley, Calif).

RESULTS

There were 14 women and 11 men, with a median age of 33 years (range, 9–74 years). Fifteen patients (60%) were aged less than 30 years. The tumor was located in the trachea in 1 patient, the right lung in 14 patients (56%), and the left lung in 10 patients (44%).

Clinical Characteristics

A history of cancer was found in 5 patients (20%), including breast cancer ($n = 1$), testis cancer ($n = 1$), retroperitoneal fibrosarcoma ($n = 1$), bronchioloalveolar carcinoma ($n = 1$), and vocal cord carcinoma ($n = 1$) (Table 1). Thirteen patients (52%) were symptomatic and presented with cough ($n = 5$), fever ($n = 4$), dyspnea ($n = 3$), dysphagia ($n = 1$), or chest pain related to T1 nerve root invasion ($n = 1$). The remaining patients were asymptomatic, and their lung tumors were discovered incidentally on routine chest radiographs.

Two patients were referred after an incomplete resection at a different institution. One of them (patient number 10), an 11-year-old child, had undergone operation 3 times

TABLE 1. Characteristics of patients who underwent complete surgical resection of inflammatory pseudotumor

Patient No.	Age, y	Sex	History of cancer	Size (cm)	Recurrence	Site of primary lesion	Cerebral metastasis	Follow-up (mo)
1	26	M		3.5	No	LLL	No	369
2	26	F		3	No	LLL	No	26
3	13	M		4	No	RUL, ML	No	244
4	29	F		4	No	RP, carina	No	223
5	20	F		7	No	RUL	No	152
6	19	F		1	No	Trachea	No	123
7	18	M		2	No	RUL	No	113
8	48	F		2.5	No	LUL, LPA	No	96
9	26	F		3.2	No	LLL	No	104
10	11	M		13	No	Extensive LP	No	0
11	36	F	Breast cancer	1.5	No	LUL	No	100
12	22	M	Testicular cancer	1	No	RUL	No	97
13	39	M		1.5	No	ML	No	62
14	29	F		2.2	No	RUL	No	53
15	12	M	Fibrosarcoma	1	Yes	RUL	No	27
16	54	F		5	No	RUL, ML	No	38
17	55	M	Bronchoalveolar cancer	4	No	Pancoast	No	12
18	9	F		2	No	RUL	No	20
19	40	F		2.5	No	RP, carina	No	15
20	30	M		3.5	No	LUL	No	20
21	70	F		3	No	RPA	No	60
22	58	M		0.6	No	LLL	No	19
23	74	M	Vocal cord cancer	5	No	Pancoast	No	25
24	19	F		11	No	RP	Yes	7
25	37	F		1.5	No	LMB	No	4

LLL, Left lower lobectomy; LUL, left upper lobectomy; ML, middle lobectomy; RP, right pneumonectomy; RUL, right upper lobectomy; RPA, right pulmonary artery; LPA, left pulmonary artery; LMB, left main bronchus; extensive LP, left sleeve pneumonectomy with esophageal, aortic arch, and right pulmonary artery resection.

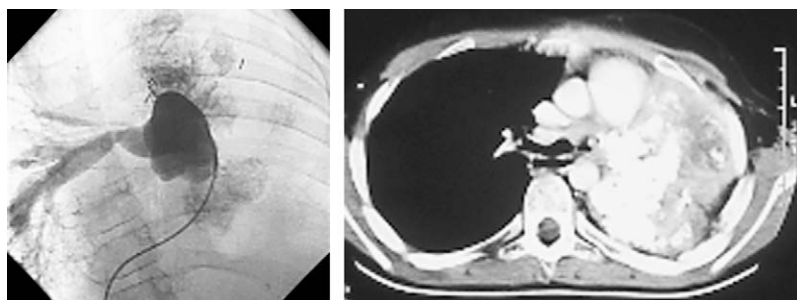


FIGURE 1. Angiography and chest computed tomography of patient 10 with an invasion of the left pulmonary artery, a tumoral stenosis of the right pulmonary artery, and an involvement of the oesophagus who had undergone operation 3 times before with uncompleted resection for an invasive mediastinal form.

before with incomplete resection margins for an invasive mediastinal variant (Figure 1). The residual mass had been managed with postoperative radiation therapy (45 Gy) and steroid therapy. This treatment failed to control the tumor locally and resulted in recurrence with invasion of the left atrium, pulmonary artery with a severe pulmonary hypertension, aortic arch, carina, and esophagus (Figure 1). He was then referred to the Marie Lannelongue Hospital for a salvage procedure.

The other patient (patient number 17) was referred for a right lung tumor invading the right thoracic inlet (first rib, subclavian artery and vein, and T1 nerve root) with chest pain. After failure to obtain negative surgical margins, adjuvant radiation therapy (50 Gy) was given at an outside institution. The patient was then referred 6 months later to our department because of further growth of the residual tumor mass.

Preoperative Workup

A pulmonary mass (>4 cm) was found in 7 patients and calcified in 6 of them (Figure 2). On bronchoscopy, we found an endobronchial lesion in 2 patients. Sixteen patients presented with a solitary nodule or lobar infiltrate. One patient (number 24) was found with 2 brain metastases during the preoperative workup.

Preoperative histologic diagnosis was obtained in 8 patients by surgical (n = 2), bronchoscopic (n = 4), or computed tomography scan-guided (n = 2) biopsy. In the remaining 17 patients, a frozen section was performed during surgery to obtain a diagnosis. In 12 of the 17 patients, the pathologist could make a definitive diagnosis of pulmonary IPT based on the frozen section. The remaining 5 patients were further reviewed before a final decision was issued.

Surgical Resection

A complete resection was always performed. The extent of resections included wedge resection (n = 7), lobectomy (n = 6), sleeve arterial lobectomy (n = 1), lobectomy ex-

tended to the thoracic inlet (n = 2), bilobectomy (n = 2), pneumonectomy with brain metastasectomy (n = 1), sleeve pneumonectomy (n = 2), sleeve main bronchus or tracheal resection (n = 2), sleeve left arterial pneumonectomy (n = 1), and sleeve pneumonectomy with esophageal, aortic arch, and right pulmonary artery resection and replacement (n = 1). Resection was performed using a cardiopulmonary bypass in 2 patients. For tumors invading the thoracic inlet, an anterior approach as described by Darteville and colleagues¹⁷ was performed in 1 patient and a posterior approach as described by Shaw and colleagues¹⁸ was performed in 1 patient.

Histologic Findings

The mean tumor diameter was 3.5 cm (range, 0.6–13 cm). The histologic types were myofibroblastic inflammatory tumor (n = 16) when there was a spindle-cell component expressing smooth muscle actin, fibrous histiocytic type (n = 8) when the spindle cell did not express smooth muscle actin, and unclassified (plasma cell granuloma, n = 1). Mycobacterial and fungal infections were ruled out in all cases by culture.

Immunohistochemically, reactions of monoclonal antibodies against cytokeratins (AE 1–3), CD34, S100 protein, and anaplastic lymphoma kinase-1 were always negative. However, tumor reactivity for smooth muscle actin (1A4 clone) was exhibited in 18 patients (72%).

Operative Mortality and Morbidity

Postoperative mortality and morbidity rates were 4% and 8%, respectively. The patient who underwent sleeve pneumonectomy with esophageal, aortic arch, and right pulmonary artery resection and replacement died postoperatively of multiorgan failure. Postoperative complications occurred in 2 other patients, including a pneumonia that was managed with intravenous antibiotics and an empyema without bronchopleural fistula that was managed by a chest tube. In all patients, the median length of stay was 11 days (range, 7–25 days). A blood cell transfusion was required in 2 patients.

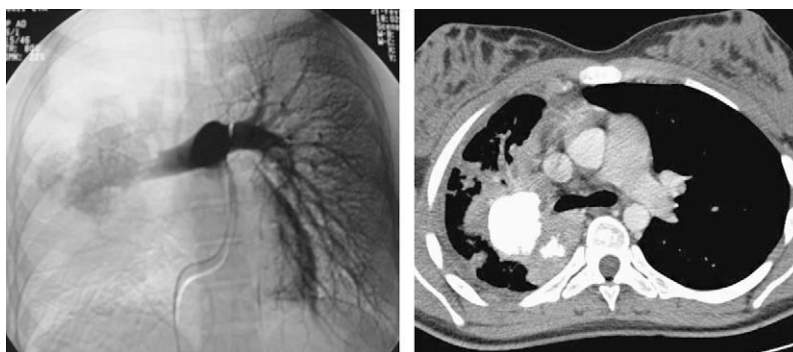


FIGURE 2. Angiography and chest computed tomography of patient 24 with an invasion of the right pulmonary artery with a calcified tumor of the right lung. This patient also presented cerebral localization with the same histology of IPT.

The patient was then referred 6 months later to our department because of further growth of the residual tumor mass.

Survival

According to the policy for these lesions at the Marie Lannelongue Hospital, no adjuvant treatment was given on discovery. Patients were closely followed at 3-month intervals for 1 year and then annually. Every visit included a physical examination and a computed tomography scan. No patients were lost to follow-up. During the study, 1 patient died of an extensive sarcomatous recurrence 27 months after surgery. The remaining 23 patients are currently alive without evidence of recurrence. With a median follow-up of 80 months (range, 4–369 months), overall 5- and 10-year disease-free survivals were 89%, respectively (Figure 3). Univariate analysis demonstrated that the only risk factor for poor outcome was the need for reoperation for tumor recurrence (5-year survival, 0% vs 96%, $P = .005$). However, the significance of this statistical test should be considered with precaution because of the small number of patients. The young patient (number 24) with cerebral metastasis is currently alive without cerebral recurrence.

DISCUSSION

Thoracic IPTs typically present as small peripheral tumors. They constitute 0.7% to 1% of all tumors of the lung.^{15,16} The incidence of these tumors is 0.04%.⁴ At the Marie Lannelongue Hospital they represented 25 of 44,019 thoracic procedures between 1974 and 2007, an incidence of 0.05%. In our series, 15 patients (62%) were aged less than 30 years. According to the reported literature, pulmonary IPTs are the most frequently diagnosed pediatric primary lung tumor.^{6,7,9,16,19–22} Both sexes are equally affected. No geographic or ethnic predominance has been reported. The precise cause of IPT of the lung is unknown. IPT of the lung is thought to be an uncontrolled response to tissue damage of chronic inflammation. The inciting injury would seem to be related to a pulmonary infection.²³ No genetic predisposition or environmental exposure has

been linked to this disease. A history of a pulmonary infection has been reported. In our series, 5 patients (21%) had a history of cancer. This trend has not been described in previous reports.

Fifty percent of patients were asymptomatic in our series, which is comparable to other reports. The tumor is frequently discovered on routine chest radiograph and presents as a solitary calcified mass with no evidence of malignancy.¹³ As in the more recent patients of the current series, the positron emission tomography imaging is positive with tumoral fixation.²⁴

In 1984, Spencer²⁵ proposed to use the term “plasma cell histiocytoma complex” IPT and pulmonary histiocytofibroma as different stages in the progression of this disease. He presented 27 pulmonary lesions, of which 2 progressed into a sarcomatous variant. A new histologic classification of pulmonary IPTs was presented by Gal and colleagues in 1994.² They classified IPT as part of a continuum of pulmonary fibrohistiocytic lesions that ranged from a typical inflammatory lesion without any evidence of malignancy to a malignant histiocytobroma. The spectrum included lesions such as benign fibrohistiocytoma, fibrohistiocytic type, malignant fibrous histiocytoma, and pulmonary IPT. Furthermore, they defined a subvariant of pulmonary IPT

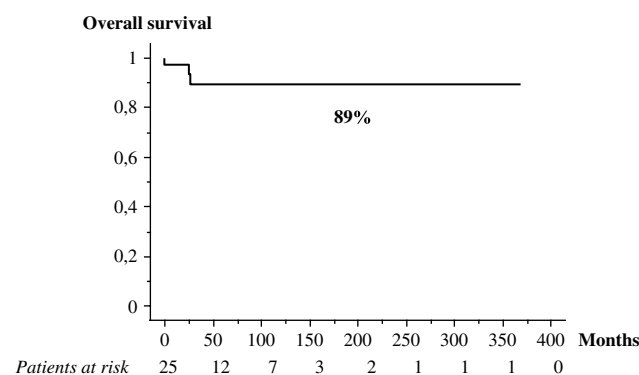


FIGURE 3. Overall survival of the 25 patients after resection of pseudo-inflammatory tumor calculated with the Kaplan–Meier method.

referred to as IPT fibrohistiocytic type. Cerfolio and colleagues⁴ have divided pulmonary IPTs into 2 groups: noninvasive and invasive IPT. They suggested an immunohistochemical study to distinguish a pulmonary IPT from a spindle-cell carcinoma or anaplastic lymphoma.

IPT in pediatric patients has a high risk of locoregional extension. This invasive form is most often described in young boys aged approximately 10 years.⁴ The mediastinal invasion can reach the contralateral lung and result in severe mediastinal sclerosis, as we discovered in patient number 10.

IPTs of the lung have a high potential for local invasion. Thus, vascular invasion,²⁴ left ventricle invasion,²⁶ and vertebral invasion have been reported.²⁵ Such infiltrating and invading forms represent approximately 20% of thoracic IPTs in children.⁴ In 6 of our cases, the local invasiveness of the pulmonary IPT required a complex debulking operation: Two tumors invaded the carina, 2 tumors invaded the thoracic inlet, 1 tumor invaded the right pulmonary artery, and 1 tumor involved the mediastinum. Metastases are described for abdominal IPT.¹⁰ Patient number 24 in our series presented with 2 lesions: a right calcified lung tumor and a cerebral metastasis. The histologic examination was the same for both lesions. Metachronous cerebral metastasis has also been described by Melloni and colleagues²⁷ 1 year after surgery; however, we could not find synchronous cerebral metastasis for IPT of the lung in the literature. For patient number 15, we found 2 tumor deposits on histologic examination: One part was IPT, and 1 part was low-grade sarcoma. This young boy had an invasive form, and the recurrence of the sarcomatous portion was fatal.

Chromosomal abnormalities, such as translocations (t [1;2] [q21;p23]) and deletions (del [4] [q27]), have been described in up to 72% of cells, previously examined in a 30-year-old woman with a middle-lobe IPT.²⁸ In contradistinction to those who regard these lesions as benign, Snyder and colleagues²⁸ described them as potentially sarcomatous lesions more than a simple inflammatory reaction. Biselli and colleagues¹² studied the ploidy of 9 pediatric IPTs. In their study, 3 patients presented with a thoracic component. Two of their patients had an invasive form, and 1 patient had a metastatic lesion (these patients presented with a mesenteric lesion, colic lesion, and pulmonary lesion [a brain metastasis subsequently developed in this patient]). In these 3 cases, there were some chromosomal abnormalities. For the patient with metastasis, the primitive lesion and metastasis had the same abnormalities but they were more pronounced in the metastasis.

The treatment of choice for IPT of the lung is surgery.^{4,7,21,22,27} For small peripheral tumors, it is now recommended to perform a wedge resection and lobectomy when the lesion is central. In other cases, when there is an invasion of the chest wall, cervico-thoracic junction, main bronchus or the carina, and diaphragm, an aggressive approach with an en bloc resection is necessary to ensure long-term survival.

Steroid therapy has been used successfully by Bando and colleagues²⁹ for 2 patients as a primary mode of treatment. However, in their report, the follow-up was short and no information on late recurrence was provided. This treatment has also been used for IPT with a contraindication for surgical resection either because of invasive presentation deemed unresectable or functionally inoperable patients.³⁰⁻³⁴ The results of corticosteroid treatment are widely variable, ranging from inefficacy to complete regression.^{6,22,29-31}

Six patients have been treated with chemotherapy;^{4,21,32,33} only 1 of them, treated with corticosteroids associated with 3 cycles of chemotherapy combining doxorubicin, cyclophosphamide, vinblastine, and bleomycin, had a complete regression of the tumor with a follow-up at 5 years.²¹ In 2 major recurrences, Cerfolio and colleagues⁴ described a failure of chemotherapy (agents not mentioned). Imperato and colleagues³³ believe that in rare cases in which the lesion is locally aggressive and surgically unresectable or resectable only with major morbidity, radiation therapy can be an effective alternative. Currently, the recommended treatment is 4000 to 4500 rads given in 180 to 200 rad fractions, with the fields being carefully tailored to tumor volume to minimize the dose to the surrounding normal tissue.³³⁻³⁵ Because the results of chemotherapy and radiation therapy are also widely variable, primary surgical treatment for most patients is still recommended, especially in young patients who can tolerate extended surgery. In case of contraindication to surgical resection, we recommend steroid or chemotherapy rather than radiation therapy to avoid local side effects.

CONCLUSIONS

IPT of the lung is rare but should be considered in the differential diagnosis of a calcified solitary tumor in a young patient. Despite the evidence that most of these tumors show a benign clinical course, it is clear that these tumors can have mediastinal invasion, local recurrences, metastases, and sarcomatous degeneration. These invasive presentations suggest that in reality they are malignant neoplasms. We can also define 2 classes of tumor: peripheral tumors easily treated with wedge resection or lobectomy and central tumors necessitating a resection that can be extended to the tracheobronchial tree or adjacent organs. These tumors should be removed or they can progress to malignant forms, such as mesenchymal low-grade tumors. Once a complete resection is performed for pulmonary IPTs, there is rarely evidence of recurrence despite long-term follow-up. A complete resection should always be attempted because it was associated with a 10-year survival of 89% in our series.

References

1. Pettinato G, Manivel JC, Insabato L, De Chiara A, Petrella G. Plasma cell granuloma (inflammatory pseudotumor) of the breast. *Am J Clin Pathol*. 1988;90: 627-32.
2. Gal AA, Koss MN, McCarthy WF, Hochholzer L. Prognostic factors in pulmonary fibrohistiocytic lesions. *Cancer*. 1994;73:1817-24.

3. Agrons GA, Rosado-de-Christenson ML, Kirejczyk WM, Conran RM, Stocker JT. Pulmonary inflammatory pseudotumor: radiologic features. *Radiology*. 1998;206:511-8.
4. Cerfolio RJ, Allen MS, Nascimento AG, Deschamps C, Trastek VF, Miller DL, et al. Inflammatory pseudotumors of the lung. *Ann Thorac Surg*. 1999;67:933-6.
5. Maier HC, Sommers SC. Recurrent and metastatic pulmonary fibrous histiocytoma/plasma cell granuloma in a child. *Cancer*. 1987;60:1073-6.
6. Sakurai T, Kamada H, Yasuoka Y, Furuya N. [Inflammatory pseudotumor arising in the ethmoid sinus: a case report]. *Nippon Jibiinkoka Gakkai Kaiho*. 2005;108:806-9.
7. Urschel JD, Horan TA, Unruh HW. Plasma cell granuloma of the lung. *J Thorac Cardiovasc Surg*. 1992;104:870-5.
8. Hoer J, Steinau G, Fuzesi L, Gunawan B, Schumpelick V. Inflammatory pseudotumor of the diaphragm. *Pediatr Surg Int*. 1999;15:387-90.
9. Coffin CM, Humphrey PA, Dehner LP. Extrapulmonary inflammatory myofibroblastic tumor: a clinical and pathological survey. *Semin Diagn Pathol*. 1998;15:85-101.
10. Meis JM, Enzinger FM. Inflammatory fibrosarcoma of the mesentery and retroperitoneum. A tumor closely simulating inflammatory pseudotumor. *Am J Surg Pathol*. 1991;15:1146-56.
11. Sciot R, Dal Cin P, Fletcher CD, Hernandez JM, Garcia JL, Samson I, et al. Inflammatory myofibroblastic tumor of bone: report of two cases with evidence of clonal chromosomal changes. *Am J Surg Pathol*. 1997;21:1166-72.
12. Biselli R, Ferlini C, Fattorossi A, Boldrini R, Bosman C. Inflammatory myofibroblastic tumor (inflammatory pseudotumor): DNA flow cytometric analysis of nine pediatric cases. *Cancer*. 1996;77:778-84.
13. Ishida T, Oka T, Nishino T, Tateishi M, Mitsudomi T, Sugimachi K. Inflammatory pseudotumor of the lung in adults: radiographic and clinicopathological analysis. *Ann Thorac Surg*. 1989;48:90-5.
14. Maples MD, Adkins RB Jr, Graham BS, Dao AH, Scott HW Jr. Pseudotumor of the lung. *Am Surg*. 1985;51:84-8.
15. Coffin CM, Dehner LP, Meis-Kindblom JM. Inflammatory myofibroblastic tumor, inflammatory fibrosarcoma, and related lesions: an historical review with differential diagnostic considerations. *Semin Diagn Pathol*. 1998;15:102-10.
16. Matsubara O, Tan-Liu NS, Kenney RM, Mark EJ. Inflammatory pseudotumors of the lung: progression from organizing pneumonia to fibrous histiocytoma or to plasma cell granuloma in 32 cases. *Hum Pathol*. 1988;19:807-14.
17. Dartevelle PG, Chapelier AR, Macchiarini P, Lenot B, Cerrina J, Ladurie FL, et al. Anterior transcervical-thoracic approach for radical resection of lung tumors invading the thoracic inlet. *J Thorac Cardiovasc Surg*. 1993;105:1025-34.
18. Shaw RR, Paulson DL, Kee JL. Treatment of superior sulcus tumor by irradiation followed by resection. *Ann Surg*. 1961;154:29-40.
19. Dehner LP. Inflammatory myofibroblastic tumor: the continued definition of one type of so-called inflammatory pseudotumor. *Am J Surg Pathol*. 2004;28:1652-4.
20. Berman M, Georgiou GP, Schonfeld T, Feinmesser M, Horev G, Vidne BA, et al. Pulmonary inflammatory myofibroblastic tumor invading the left atrium. *Ann Thorac Surg*. 2003;76:601-3.
21. Copin MC, Gosselin BH, Ribet ME. Plasma cell granuloma of the lung: difficulties in diagnosis and prognosis. *Ann Thorac Surg*. 1996;61:1477-82.
22. Bahadori M, Liebow AA. Plasma cell granulomas of the lung. *Cancer*. 1973;31:191-208.
23. Frey A, Eichfeld U, Schubert St, Friedrich T, Schonfelder M. [Inflammatory pseudotumor of the lung in hilus lymph node histoplasmosis]. *Chirurg*. 1998;69:1101-4.
24. Slosman DO, Spiliopoulos A, Keller A, Lemoine R, Besse F, Couson F, et al. Quantitative metabolic PET imaging of a plasma cell granuloma. *J Thorac Imaging*. 1994;9:116-9.
25. Spencer H. The pulmonary plasma cell/histiocytoma complex. *Histopathology*. 1984;8:903-16.
26. Kelly SJ, Lambie NK, Singh HP. Inflammatory myofibroblastic tumor of the left ventricle in an older adult. *Ann Thorac Surg*. 2003;75:1971-3.
27. Melloni G, Carretta A, Ciriaco P, Arrigoni G, Fieschi S, Rizzo N, et al. Inflammatory pseudotumor of the lung in adults. *Ann Thorac Surg*. 2005;79:426-32.
28. Snyder CS, Dell'Aquila M, Haghighi P, Baergen RN, Suh YK, Yi ES. Clonal changes in inflammatory pseudotumor of the lung: a case report. *Cancer*. 1995;76:1545-9.
29. Bando T, Fujimura M, Noda Y, Hirose J, Ohta G, Matsuda T. Pulmonary plasma cell granuloma improves with corticosteroid therapy. *Chest*. 1994;105:1574-5.
30. Shirakusa T, Kusano T, Motonaga R, Eimoto T. Plasma cell granuloma of the lung—resection and steroid therapy. *Thorac Cardiovasc Surg*. 1987;35:185-8.
31. Doski JJ, Priebe CJ Jr, Driessnack M, Smith T, Kane P, Romero J. Corticosteroids in the management of unresected plasma cell granuloma (inflammatory pseudotumor) of the lung. *J Pediatr Surg*. 1991;26:1064-6.
32. Kirk VG, McFadden S, Pinto A, Boag G, Sigalet DL. Leiomyoma of the esophagus associated with bronchial obstruction owing to inflammatory pseudotumor in a child. *J Pediatr Surg*. 2000;35:771-4.
33. Imperato JP, Folkman J, Sagerman RH, Cassady JR. Treatment of plasma cell granuloma of the lung with radiation therapy. A report of two cases and a review of the literature. *Cancer*. 1986;57:2127-9.
34. Hoover SV, Granston AS, Koch DF, Hudson TR. Plasma cell granuloma of the lung, response to radiation therapy: report of a single case. *Cancer*. 1977;39:123-5.
35. Shapiro MP, Gale ME, Carter BL. Variable CT appearance of plasma cell granuloma of the lung. *J Comput Assist Tomogr*. 1987;11:49-51.